REMARKS

Favorable reconsideration is respectfully requested in view of the foregoing amendments and the following remarks.

Claims 1-29 are cancelled without prejudice or disclaimer thereto.

Claims 30-41 are newly added.

In new claim 30, the limitation of "which expresses β-amyloid peptide in intestinal cells" from former claim 19 is deleted. Further, the limitation "whereby the concentration of TGF-β in the blood of the subject is reduced" is incorporated into new claim 30. The basis for the amendment is found on page 3, lines 26 to 27, of the specification.

New claim 31 is based on the limitation "an adeno-associated virus vector which expresses β-amyloid peptide in intestinal cells" in former claim 19.

New claims 32 to 41 correspond to former claims 20 to 29.

On pages 2-15 of the Office Action, claims 19-21 and 29 were rejected under 35 U.S.C. § 103(a) as being unpatentable over Huston et al. (US 2005/0255113) in view of Kuwako et al. (Kuwako et al., *Brain Res Mol Brain Res.* 107(2):166-75, 2002), Milton et al. (WO 2002/36614) (of record), and Findeis et al. (US 5,854,204).

This rejection is moot as these claims have been cancelled without prejudice or disclaimer thereto. Furthermore, regarding new claim 30, the presently claimed invention is directed to a method for treating Alzheimer's disease, comprising administering an adeno-associated virus vector in a therapeutically effective amount to a subject whereby the concentration of TGF-β in the blood of the subject is reduced, and wherein the adeno-associated virus vector comprises DNA encoding a β-amyloid peptide and DNA encoding a signal peptide capable of extracellularly secreting said β-amyloid peptide, in an operative form.

It has been reported that TGF-β promotes Alzheimer's disease-related pathological changes such as cerebrovascular amyloid deposition and microvascular degeneration (see page 2, lines 21 to 29 of the specification). Thus, the method of the claimed invention capable of reducing TGF-β in the blood is advantageous in reducing the progress of cerebrovascular amyloid deposition and microvascular degeneration associated with Alzheimer's disease (see page 3, lines 27 to 29).

On the other hand, the Examiner rejected the former claims for obviousness in view of Huston et al. (US 2005/0255113), Kuwako et al. (Mol Brain Res, 107(2):167-75,2002), Milton et al. (WO2002/36614) and Findeis et al. (US 5,854,204). However, these citations fail to disclose or teach a reduction in the concentration of TGF-β in the blood of a subject by administering an adeno-associated virus vector comprising DNA encoding a β-amyloid peptide and DNA encoding a signal peptide capable of extracellularly secreting the β-amyloid peptide, in an operative form.

Thus, Applicants contend that those skilled in the art could not have arrived at the method of the present invention on the basis of these citations. Thus, Applicants respectfully suggest that this rejection is untenable as applied to the newly added claims and therefore should be withdrawn.

In view of the foregoing amendments and remarks, it is respectfully submitted that the present application is in condition for allowance and early notice to that effect is hereby requested.

If the Examiner has any comments or proposals for expediting prosecution, please contact the undersigned attorney at the telephone number below.

Respectfully submitted,

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By /William R. Schmidt, II/

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